THESIS ON DYSPNOEA

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D Y S P N O E A

Dyspnoea is one of the symptoms which the physician is most frequently called upon to treat in e-. mergency. Being only a symptom and one occurring in very many different diseases, it is extremely important and to thoroughly understand its various forms and the fundamental cause in each form; without which knowledge we cannot hope to treat the condition successfully.

The word Dyspnoea, from the Greek, δυς , corresponding to the Latin"difficilis", has a wider meaning than our "difficult", and means something wrong or defective, something abnormal.

I propose discussing the mechanism of dyspnoea in general, and the special mechanism and distinctive characters, together with the pathology of the various forms of Dyspnoea in particular.

It is essential at the outset to familiarize ourselves with normal respiration.

Efficient, easy and pleasurable respiration depends on the maintenance of the normal proportion between the blood and the air supplied to the lungs, and upon the absolute and relative quantities in which they are constantly renovated- any derangement

of these proportions at once produces dyspnoea.

The Physical Mechanism of Normal respiration entails:-

I. A certain range of movement of the chest.

II. A certain rate of breathing.

III. A certain ratio of inspiration and expiration.

IV. Certain active moving powers.

It is in the disturbance of the healthy standard of these conditions that dyspnoea essentially consists, and the varieties of these disturbances are characteristic of the different forms of dyspnoea.

We will now consider these four conditions.

I. The range of movement of the chest. The average amount of air moved into and out of the chest at an ordinary respiration is twenty cubic inches according to Hutchinson, this is called tidal air.

II. A certain rate of breathing. In the adult the number of respirations varies from 16 to 24 per minute, the average according to Foster being 17.

III. A certain ratio of inspiration and expiration. When respiration proceeds quietly and regularly there is no pause between inspiration and expiration, and inspiration is to expiration as 3 is to 7 according to Rutherford,

as 10 is to 12, according to Foster.

IV. Certain active moving powers. These are partly vital and partly physical, partly consisting of nervous and muscular force and partly of elastic recoil. A slight allowance must also be made for the influence of gravity. It is necessary to determine the manner in which these several movements are carried out.

At the end of expiration there is a condition of equilibrium between two forces, the elasticity of the lungs tending to reduce still further the capacity of the chest, and that of the costal cartilages opposing any further reduction of chest capacity; that this is so is proved by the fact that when the pleura is opened immediately after death, air enters and the lung collapses, while at the same time the chest wall expands somewhat.

The act of inspiration is now started and the action of the inspiratory muscles is a third force disturbing the previously existing equilibrium. At the commencement of inspiration the inspiratory muscles have acting against them the elasticity of the lungs and with them the elasticity of the costal cartilages . A very slight expansion of the chest brings the costal cartilages to that position which

they would assume and rest at were they at any time freed from the effect of the elastic recoil of the lungs, and beyond this the inspiratory muscles have two factors to overcome, that of the chest wall and that of the lungs, both opposing and with a rapidly increasing resistance, the inspiratory distension of the chest. It is against these two forces that the inspiratory muscles have to work in distending the chest. The costal parieties offer much greater resistance to the inspiratory muscles than do the lungs.

We must now study the action of another great muscle of inspiration, namely, the Diaphragm. During inspiration it descends by the flattening of its arch, thereby elongating the chest from above downwards, and according to Brücke also increasing the transverse diameter of the lower part of the chest. It has two elastic forces opposed to its descent, acting on its opposite surfaces, the elasticity of the lungs on its upper surface tending to draw it upwards, and the elasticity of the abdominal contents and walls exercising a pressure on its under surface, we have thus muscular contractility opposed to elastic resistance, as was the case with the costal parities, with these differences, that whereas

in the former the two elastic resistances (lungs and abdominal contents and walls) act all the time in the same direction, in the latter the ribs act first concurrently and then antagonistically; also that the elastic resistance of the abdominal walls and contents increases far more slowly than that of the costal cartilages.

A sufficient quantity of air having now been inspired, there is no further need for enlarging the capacity of the chest, and muscular action ceases. Expiration now commences. The elastic forces of the ribs and costal cartilages, being freed from the stretching power of the inspiratory muscles, the lungs at once recoil.

The muscular contraction of the diaphragm having ceased, the elastic contents and walls of the abdomen press up the diaphragm, the elastic recoil of the lungs at the same time exercises traction on both costal and diaphragmatic surfaces, and so the air which was, so to speak, labouriously by muscular action drawn into the chest, is easily expelled by elastic recoil. The important point which all this brings prominently before us, is that inspiration is a muscular action brought about against opposed forces, while expiration is almost entirely non-muscular and unopposed.

Some authorities affirm that expiration is actively assisted by the internal intercostal muscles and the triangularis sterni, but the muscular power brought about by these muscles must be so slight that it may be disregarded. Probably the abdominal muscles also contract during expiration and hasten that act.

We might here note the important fact that although inspiration is muscular and expiration non-muscular, in forced respiration the muscles of forced expiration are much more powerful than those of forced inspiration, as is shown by a mercurial manometer, a forced expiration can support from 3 to 4 inches of mercury, a forced inspiration is only about half as powerful.

Gravity plays a slight and unimportant part in the act of respiration. The weight of the costal parities opposes inspiration whether standing or lying. The action of the weight of the abdominal contents depends upon the position of the body, opposing the descent of the diaphragm in the horizontal position and so telling in favour of expiration, while in the vertical position it will favour inspiration by favouring the descent of the diaphragm; so the abdominal contents favour inspiration in

standing and expiration in lying.

The influence of atmospheric pressure may be entirely disregarded as it exerts an equal pressure against inspiration and expiration.

So much for the physical mechanism of respiration, we must now briefly study its nervous mechanism.

The respiratory movements are involuntary, not only do they occur during unconsciousness, but even after removal of all the parts of the brain above the medulla oblongata.

All the carefully co-ordinated muscular contractions which are involved in the acts of inspiration and expiration are brought about by co-ordinated nervous impulses descending along efferent nerves from a co-ordinating nervous centre in the medulla. This is proved by the fact that removal or extensive injury to the Medulla alone is at once followed by the cessation of all respiratory movements, even though the rest of the nervous system, including every muscle and every nerve concerned, be left intact. There are subsidiary centres in the spinal cord which modify the respiratory impulses before they issue along the motor nerve roots.

The action of the centre is automatic and not reflex, although it may be influenced in many ways

by afferent impulses of various kinds, as well as by the action of the will or by emotion. The Respiratory centre is influenced in two ways,

(1) by nerve impulses started in various ways and reaching the centre along various nerves. Of all the automatic centres the respiratory centre is the one whose independence is most obscured by the repeated effects of afferent nerve impulses.

The Vagi are the most important afferent nerves connected with the respiratory centre, they contain fibres of two kinds, one set augmenting the action of the centre and the other set having an inhibitory effect, the most common effect being augmentation.

The Respiratory centre is a double one, consisting of an inspiratory centre and an expiratory centre, and afferent impulses reaching the medulla which augment or inhibit the one necessarily inhibit or augment the other. The Inspiratory centre is the dominant and most responsive one. The Respiratory centre consists of two lateral halves which normally work in unison and yet may be made to work independently; if the medulla be carefully divided in the middle line, respiration may go on in quite a normal fashion.

Next to the Vagi the nerve most closely connected with the respiratory centre is the 5th nerve,

branches of which guard the nasal respiratory channels. The motor nerves of ordinary inspiration are the phrenics to the diaphragm, the intercostal nerves, and the inferior laryngent nerves to the crico-arytenoid muscles which open the glottis. The motor nerves of expiration are the lumbar and sacral nerves to the muscles of the abdomen and lower part of back.

(2) The Respiratory centre is affected by the quality of the blood distributed to the medulla, in respect of its respiratory changes, more than in any other way; the effect is produced by the direct action of the blood on the medullary respiratory centre itself, and not reflexly by stimulating the peripheral ends of afferent nerves in various parts of the body. This is proved by cutting away the brain above the medulla, dividing the spinal cord below the phrenic nerves, and dividing all the nerves connected with the medulla and cervical part of the spinal cord except the phrenics, so that no afferent nerves can influence the respiratory centre, deficient oxygenation of the blood under these circumstances produces dyspnoea, the stimulation of the respiratory centre must be direct and due to the deficiency of oxygen in the respiratory centre

itself, as the phrenics, which are the only nerves left, are efferent nerves.

It is the lack of oxygen, which plays the principal part in developing the abnormal respiratory movements, and not the excess of Carbonic acid, as has been proved by numerous experiments. Conditions of the blood other than variations in the amount of oxygen and carbonic acid may materially affect the working of the respiratory centre.

Haldane, Smith, Lorrain Smith have proved the opposite.

Having now discussed these parts of the normal process of respiration which it is important to understand in studying its various derangements, we will turn our attention to Dyspnoea. It is extremely difficult to formulate a perfectly satisfactory classification, and all classifications of Dyspnoea must be more or less imperfect owing to the fact that more than one cause is in operation in nearly every case.

It will be seen from the facts already stated that in every form of Dyspnoea except the rare and unimportant cases due to direct irritation of the respiratory centre in the medulla by tumours, inflamatory deposits, etc., there is always one constant condition present, namely, an abnormal state of the blood stimulating the respiratory centre. This is a point which I think has not been suffici-

ently emphasized before, and it gives us a sound basis for a good classification.

With this in mind I purpose dividing Dyspnoea into four classes:-

I. Dyspnoea due to interference with the access of air to the alveolar capillaries of the lungs.

(a) obstruction in the air passages, the lung substance being healthy.

(b) General or partial spoiling of the lung substance, the air passages being healthy.

(c) Interference with the normal active moving powers.

(1) Loss of elasticity.

(2) Loss of muscular power.

II. Dyspnoea due to interference with the circulation through the lungs.

(a) Heart disease.

(b) Obstruction of the pulmonary artery or its branches by emboli.

III. Dyspnoea due to an abnormal state of the blood.

(a) Anaemia

(b) An impure altered state of the blood.

IV. Dyspnoea of central nerve origin.

I. Dyspnoea due to interference with the access of air to the alveolar capilaries of the lungs.

(a) Obstruction in the air passages, the lung substance being healthy.

When the obstruction is above the smaller bronchi the Dyspnoea is inspiratory in character and the inspiration frequently noisy, there is often a great amount of respiratory distress, but the breathing is frequently not quickened, indeed it may be preternaturally slow, the stream of air being so narrow that it takes a long time to get in. The range of movement is often small, there is frequently an indrawing of the thoracic wall during inspiration, most marked in the supra clavicular regions and intercostal spaces. Both inspiration and expiration are greatly lengthened, but inspiration much more so than expiration.

On auscultation the breath sounds are feeble; if the obstruction is in the larynx or trachea a stridor is heard, if in a large bronchus, a snoring rhonchi.

An important distinction between obstruction in the larynx and obstruction lower down, first pointed out by Gerhardt, is that in the former

during inspiration there is a rapid and extensive movement of the larynx downwards, which does not occur when the obstruction is lower down.

The reasons that in obstruction of the upper respiratory passages the Dyspnoea is inspiratory are, I believe:-

(1) That before Dyspnoea is produced the obstruction is so great that it comes to be a question which act is the more powerful - forced inspiration or forced expiration, we have seen that the latter is two or three times as powerful as the former, and so the greatest difficulty occurs during inspiration.

(2) The most important reason is that during inspiration the atmospheric pressure becomes much less below the point of obstruction than at it, and so a suction action takes place which leads to an indrawing of the walls of the air passage at that point, and so during inspiration there is actually greater obstruction to the passage of air than during expiration.

When the obstruction is in the bronchioles the Dyspnoea is always expiratory, as was first noticed by Riegal. I would suggest as an explanation of this, that, as we have seen the act of inspiration to be entirely muscular, while that of expiration

ordinary

is non-muscular, when an obstruction occurs telling equally both ways, a simple increase in the muscular effort which is constantly in action and constantly being called into greater action, as in physical exertion, is not noticed, whereas during expiration when air has to be expelled from the lungs against some obstruction, muscular force is required, which under normal conditions, even during physical exertion, there being no interference with the entrance and exit of air, is not required, then this act is felt to be laboured and an entirely unusual condition is experienced. In expiratory Dyspnoea then the obstruction or lesion is always in the bronchioles or alveoli, which is an important fact to bear in mind.

The two diseases causing Dyspnoea which come under our present heading are Bronchitis and Asthma.

In Bronchitis when Dyspnoea occurs it is usually due to spasm of the bronchial muscles, as has been demonstrated by Professor Fraser, who on giving one of the preparations of the nitrites found that immediately the Dyspnoea was relieved and the rhonchi disappeared with extraordinary rapidity. In a certain proportion of cases of Bronchitis the dyspnoea is not thus relieved, and here it is due to the

Bronchitis assuming the capillary form, with the accumulation of mucous in most of the bronchioles obstructing the entrance of air. The dyspnoea is also increased by the pyrexia which usually accompanies the capillary bronchitis.

In Asthma the dyspnoea is also due to narrowing of the lumen of the bronchioles, how this narrowing is brought about has long been a subject of discussion. It is now generally believed to be due to spasm of the bronchial muscles, which may be brought about in very many different ways.

C.J.B.Williams first conclusively proved the existence of the bronchial muscles, and in his experiments on the lungs of oxen, dogs and rabbits he was able to cause contraction of the bronchial tubes by the application of electrical, chemical and mechanical stimuli; he proved that the muscular coat was most abundant in the smallest bronchial tubes and by sufficiently strong stimuli made them contract so much as to obliterate their lumen entirely. Branches of the Vagi are the motor nerves to these bronchial muscles.

In Asthma the spasm may be brought about in many different ways, through the nervous system by central stimulation of the vagi roots, reflexly the

through various paths. In a large number of cases the vagus with its pulmonary and gastric plexûs is the seat of the disease, in a number of cases the nervous circuit between the source of irritation and the seat of the resulting muscular spasm involves other parts of the nervous system besides the vagus. The fifth nerve is often the afferent nerve through its branches, supplying the mucous membrane of the nose, as in nasal polypi, etc. In the so-called dyspeptic asthma the nerve impulse passes through the gastric branches of the vagi, and is reflected by the motor filaments of the pulmonary plexûs. The branches of the pulmonary plexûs, anterior and posterior, form a net-work round the bronchial tubes and contain some minute ganglia, when the spasm is direct as from dust, etc., inhaled, possibly by irritation of Leyden's Crystals, it may be induced by reflex action through these small ganglia or through the pulmonary plexûs, though it soon extends deeper into the nervous system, involving the vagi and causing a motor effect on the thoracic muscles through the upper cervical, dorsal and phrenic nerves.

In a large number of cases of Asthma it is impossible to find a source of nerve irritation; it has long been observed that many of these cases

have a gouty diathesis, and probably the spasm is brought about by the influence of the products of defective tissue metabolism circulating in the blood and lymph, there being defective elimination, and irritating nerve filaments in connection with the pulmonary plexus, or they may cause contraction of the bronchial muscles by coming directly in contact with them through the lymph stream.

The character of the dyspnoea of Asthma is, as we have said, expiratory, expiration is greatly prolonged, generally being two or three times as long as inspiration, the rate of breathing is slowed, often falling as low as 9 per minute, the range of movement of the chest is small, the chest is distended and contains a number of cubic inches of air more than normal. The active moving powers are altered, the muscles of forced inspiration and still more markedly those of forced expiration are called into play, the patient assuming the sitting posture so as to give these muscles the greatest possible purchase on the chest. On auscultation the inspiratory vesicular murmur can hardly be heard, usually a sibilus or a sonorous rhonchi replaces it, during expiration a loud wheezing sound is heard; the tone of these sounds vary according to the calibre of the bronchial tubes, the smaller ones giving rise to

high notes the larger to low notes. These rhonchi continually change their position, springing up under the listening ear and as quickly disappearing again.

Before leaving this disease it must be mentioned that another theory as to the cause of the obstruction in the bronchioles held by many German authorities and by the late Sir Andrew Clarke in this country, is, that an urticaria-like swelling of the mucous membrane of the bronchioles occurs due to vaso motor disturbance; this certainly cannot occur in those cases which are relieved by nitrites, as the action which the nitrites have in dilating the small blood vessels would increase the swelling.

Leyden thought that the crystals which he found in the sputum of asthmatical patients, by their irritation, set up reflex spasm of the bronchioles, but this is improbable, from the fact that they are found in the sputum of persons who dont suffer from asthma, or show any sign of bronchial spasm.

Having now discussed those cases in which the interference with the access of air to the lungs is due to obstruction in the air passages, we will pass on to consider the cases where it is due to

(b) General or partial spoiling of the lung substance, the air passages being healthy. Here the

remaining healthy part of the lung has to do more work than usual to compensate for the parts which are damaged. The range of movement is locally increased, the rate of breathing is quickened, more air is changed at each respiration, the normal ratio of inspiration and expiration is usually maintained. In this class we meet with the most hurried of all forms of breathing.

On auscultation the breathing is puerile or compensatory in the healthy parts of the lung. Some times very marked bronchial breathing is heard in parts, owing to the parenchyma of the lung being compressed and so the vesicular murmur is absent in such areas.

This class includes collapse of the lung from hydro, haemato, pneumo and pyo-thorax, pressure on the lungs by mediastinal or abdominal tumours, ascites, pregnancy, flatulence, etc., also acute oedema, tuberculous and cancerous infiltration of the lungs, and in pneumonia where a large extent of lung substance is involved. In most cases of lobar pneumonia, however, I believe the dyspnoea does not depend upon the loss of respiratory surface of the lung, but to the pyrexia and the absorption by the blood of ptomaines and waste products which stimulate the respiratory centre in the medulla directly,

because when the crisis is reached the rate of breathing falls pari passu with the temperature and before the exudation in the air cells could possibly have been absorbed,

The access of air to the alveolar capillaries of the lungs may also be prevented by

(C) Interference with the normal active moving powers.

(1) Loss of elasticity, this occurs typically in emphysema, the air cells having become so over-distended that they have lost their power of elastic recoil; the chest is over-distended and the range of movement is diminished, inspiration is short and quick, and is followed by a greatly prolonged expiration. This prolonged expiration (there being no obstruction of the air passages) is pathognomonic of dyspnoea dependant upon the loss of the natural contractility of the lung, and, as well as in emphysema, it occurs in cancerous and tubercular infiltrations of the lung, the normal elastic tissue being replaced by a non-elastic new deposit.

The rate of breathing is usually not increased owing to the great lengthening of expiration, there is usually very little respiratory distress. Expiration is very frequently accompanied by a wheezing sound or rhonchi, there being some obstruction in the smaller bronchioles.

(2) Loss of muscular power. In injuries of the spinal cord below the 5th cervical vertebra, all the respiratory muscles except the diaphragm are paralysed, respiration is then entirely abdominal and is very laboured. When the injury is above the origin of the phrenics death rapidly ensues. If the paralysis is of gradual onset there is a subjective feeling of want of power to breathe and the respiratory movements are shallow and hurried and soon death occurs from asphyxia.

There is a peculiar form of dysphoea due to paralysis of the diaphragm, the other muscles of respiration being unaffected, this affection was first recognised and described by Duchenne.
When at rest there is no dyspnoea and the breathing is easy; on the slightest exertion there is great

distress and the rate of breathing is very much increased, there is a sense of suffocation. On taking a deep inspiration there is a feeling as though the abdominal organs were being drawn into the chest. During inspiration when the ribs rise and the chest expands the abdominal wall becomes drawn in, during expiration it is pushed forward, thus the relation to the thoracic movements is exactly the reverse to what it normally is. Cases of this peculiar affection have been recorded by Duchenne in progressive muscular atrophy and lead poisoning; by Walshe as a sequel of diphtheria, Aran gives extension of inflammation from the peritoneum or pleura as a cause.

Far commoner than these latter affections are those in which the normal active moving powers of respiration are interfered with by pain, as in pleurisy, pleurodynia, rheumatism of the intercostal muscles, peritonitis &c.

Here the great characteristic of the dyspnoea is the limited movement of the thorax, the breathing is superficial, a small amount of air is changed at

each respiration, the range of movement is greatly diminished, the chest is kept at the greatest possible distance from inspiratory distension, often a long way on the expiratory side of the normal position of equilibrium at the end of expiration. The breathing is hurried and according to the position of the pain, the movement at that part is limited as much as possible and so the two sides of the chest may not move to the same extent, or the breathing may assume a costal or abdominal character according to the position of the pain.

II. Dyspnoea due to interference with the circulation through the lungs. The dyspnoea in this class is always due to deficient supply of oxygen to the respiratory centre in the medulla, and not, as was formerly supposed, reflexly from stimulation of the pulmonary branches of the vagi in the lungs by venous blood engorging the pulmonary capillaries. This form of dyspnoea occurs in two classes of cases. (1) Heart disease of any form where there is increased backward and diminished forward pressure, as in valvular disease, fatty heart &c., here there is a

delay of the blood in the pulmonary vessels, the red cells cannot take up a compensating amount of oxygen, and on the other hand less blood is sent over the body than usual, and so the medulla, as well as the other tissues, suffers from a deficiency of oxygen and dyspnoea is produced.

The dyspnoea is of several varieties:-

(a) Where the breathing is natural and easy until some exertion is made, which at once brings on dyspnoea, the heart is equal to its task as long as the body is at rest, but as soon as ever any strain is thrown upon it, the blood is returned to the auricles more quickly than the ventricles can get rid of it, and does not pass through the lungs sufficiently rapidly to meet the demand of the body generally for arterial blood.

(b) Here the state of matters is just a degree farther advanced, the dyspnoea is constant, the heart, even when the body is at rest, being unable to propell the blood efficiently through the lungs and body generally, and this often goes on to the condition of more or less permanent orthopnoea.

(c) The dyspnoea of Heart disease is very often paroxysmal, the paroxysms being caused by many different things, sometimes they occur early in the morning and are then due to the heart failing for want of nourishment due to long fasting; it sometimes arises from sudden over distension of the cardiac cavities, the heart failing to drive on the blood; sometimes it is due to sudden oedema of the lungs.

In the above cases the air passages are all quite free and the blood, passing through the lungs more slowly than usual, must get thoroughly oxygenated, hence the dyspnoea is not due to the respiratory centre being supplied with venous blood, but to its being supplied with a deficient quantity of arterial blood and hence with a deficient amount of oxygen; the cyanosis in these cases is of course due to the blood being delayed in the capillaries by the backward pressure until it becomes venous.

The character of the dyspnoea in all these cases is similar. The breathing is very much quickened, there is rather a breathlessness than a

difficulty of breathing, the breathing is of a panting, gasping character, a sense of oppression rather than tightness is felt, a strangling, choking feeling. The range of movement is increased, more air being changed at each respiration, both inspiration and expiration are shortened, but expiration much more than inspiration. The dyspnoea is extremely intolerant of exertion and often of the recumbent posture.

In orthopnoea the sitting posture is assumed because the muscles of forced respiration can act to greater advantage in that position, also the action of gravity favours the descent of the diaphragm in that position and so tells in favour of inspiration which is the more difficult and opposed act, there being no obstruction in the air passages. In some cases the paroxysm of dyspnoea occurs just as the patient is dropping off to sleep and it has been termed nocturnal dyspnoea, it is probably due to the fact that on going to sleep a certain amount of anaemia of the brain occurs and this diminishes the already scanty supply of oxygen to the respiratory

centre to such an extent that dyspnoea occurs and the patient wakes up gasping for breath.

(d) In some forms of heart disease there is a peculiar form of dyspnoea known as Cheyne-Stokes respiration, or ascending and descending respiration, it was first observed by Cheyne and afterwards more fully studied by Stokes and hence called by their combined names, Hippocrates seems to have noticed it in the fatal illness of Philiscus,- Galen described it as 'like a person who forgot for a time the need of breathing and then suddenly remembered it.'

In this form of dyspnoea the patient experiences considerable difficulty of breathing, or rather experiences the need for rapid and forced breathing for a few respirations, then the depth of the respirations becomes gradually less and less until they are nearly and then quite imperceptible to ordinary observation and auscultation, no cyanosis or indication of distress or 'besoin de respirer' is however to be noticed. After a duration of some seconds, a just perceptible respiratory movement is to be seen, this is succeeded by a more ample one, and so on until the

maximum of activity is reached. This peculiar form of dyspnoea is met with in many forms of heart disease, Stokes thought it only occurred in fatty heart, but this is not so, it also occurs in uraemia and cerebral affections of various kinds.- The pathology of Cheyne-Stokes Respiration has occupied a good deal of attention of late years, there having been many theories advanced to account for the phenomenon. Traube believed that the respiratory centre had its irritability so lowered by a constant deficiency of oxygen that it required a much greater amount of carbonic acid in the blood than usual to stimulate it, and so the state of apnoea occurs while this excess of carbonic acid is accumulating. The carbonic acid is at length sufficient to stimulate the respiratory centre, at first slightly, and gradually more and more until the state of dyspnoea is produced. The blood now becomes oxygenated and so the respiratory centre ceases to be stimulated and we have the stage of apnoea again. Dr. Sansom agrees with this except that he considers the lowered irritability of the respiratory centre to be due to

a paresis of the centre, which he says may be produced by reflex nerve influence, but is usually due to a direct central lesion.

Filehne believes that the vaso motor as well as the respiratory centre is concerned in the condition, that the deficiency of oxygen and excess of carbonic acid at the end of the apnoea period stimulate the vaso motor centre, leading to a contraction of all the small arteries in the body and less oxygen gets to the respiratory centre and so it is stimulated and dyspnoea occurs; the blood soon becomes oxygenated and stimulation of the vaso motor centre ceases, the respiratory centre is supplied with sufficient oxygen and apnoea occurs.

Bramwell considers that the respiratory centre is in a state of excessive irritability, being easily excited and easily exhausted, he says the inspiratory centre has a discharging and a restraining action, the discharging action being excited by venous blood and the restraining inhibited, and contrary wise by arterial blood. At the end of a period of apnoea, the blood is venous and excites both the respiratory

and vaso motor centres, which latter action by diminishing the amount of blood going to the medulla still further stimulates the respiratory centre, the centre being excessively irritable a condition of dyspnoea occurs, but being easily exhausted soon stops, but the blood first becomes arterialised and so stimulation to vaso motor and respiratory centres ceases and we have the stage of apnoea.

Biot maintains that the essential conditions are a diminution of the excitability of the respiratory centre in the medulla:- a cerebral phenomenon, having for its origin an anaemia, progressive and more or less profound:- a circulatory phenomenon. He considers that in uraemia Cheyne-Stokes respiration is due to spasm of the cerebral arteries producing anaemia of the brain. All his heart cases in which it occurred had aortic regurgitation.

Dr.Little of Dublin thought that it was due to an unequal action of the two ventricles so that the left ventricle becomes unable to propel the aerated blood and stops now and then, the blood therefore remains in the lungs, pulmonary veins and left

auricle and as it has been already fully oxygenated it no longer stimulates the respiratory centre through the pulmonary branches of the vagi, thus venous blood necessary to excite the vagi is not supplied and so respiration ceases, by degrees contraction of the left ventricle partially frees the pulmonary veins and left auricle of blood and venous blood is again sent to the lungs which stimulates the filaments of the vagi and causes respiration to begin. It will at once be seen that this theory cannot hold, as the very cause which it gives for the apnoea we now know produces dyspnoea, namely, a diminished amount of arterial blood sent to the brain, and also we have seen that venous blood produces dyspnoea by its direct action on the respiratory centre and not reflexly by stimulating the branches of the vagi.

Laycock called it 'a recurrant brief apnoea' due to a sentient paresis of the respiratory centre, not necessarily dependant on structural or other disease of the heart. Hayden added to this a diminished supply of oxygen to the respiratory centre.

It seems to me that the most satisfactory theory,

and the one which fulfills all the conditions necessary for the production of the phenomenon, is that advanced first by Traube which I have referred to.

It will be now generally conceded that in Cheyne-Stokes respiration there must be some lesion of the medulla or vagi, leading to diminished excitability of the respiratory centre or imperfect conduction of nerve impulses through the efferent branches of the vagi, this, which was first advanced as a theory, has received much strength from the observations of Lizzoni who, in two cases in which this form of respiration occurred, found on post mortem examination, in one case chronic inflammatory changes in the vagi, with blood extravisations into the lymphatic spaces of the perineurium and endoneurium, and in the other case, blood extravisations scattered through the medulla. Another very important fact which I wish to lay stress upon is that the ascending and descending respiration always occurs in diseases which we know give rise to ordinary dyspnoea, and in fact dyspnoea always preceeds the occurrence of Cheyne-Stokes respiration, very possibly where there

is not an actual lesion in the medulla, the diminished irritability of the respiratory centre is due to a long continued deficient supply of oxygen to the medulla as has been suggested by Traube and Biot. Starting with these two factors, viz, the previous existance of dyspnoea and the diminished excitability of the respiratory centre; the centre gradually becomes so torpid that it only responds to blood less oxygenated than normal. In an attack of dyspnoea the rapid free breathing thoroughly oxygenates the blood and at the same time increases the rapidity of the circulation so that the medulla as well as the rest of the body is rapidly supplied with blood that has been well oxygenated and consequently the respiratory centre is not stimulated, the respiration gradually ceases and then stops, the circulation also slows and the blood soon becomes sufficiently venous to stimulate the respiratory centre and respiration commences, first gradually and then rapidly. The few rapid and violent respirations may be explained by the perverted activity of the respiratory centre showing the irritability of exhaustion.

(2) The circulation through the lungs may also be interfered with by obstruction of the Pulmonary artery or its branches by emboli. The emboli may consist of blood clot, of fat or of air, the amount of dyspnoea produced in each case depending upon the extent to which the circulation through the lungs is interfered with.

In the case of obstruction of the pulmonary artery by blood clot, Trousseau believed that it might occur in two ways, primary in lung disease, as pneumonia, when thrombosis might occur in situ, and also by migration of a peripheral clot. Virchow maintained that it could only occur in the latter way.

Beverley Robinson divided pulmonary embolism into three forms.

(a) The sudden fatal, in which the main trunk or both divisions of the pulmonary artery are plugged. Here there is intense dyspnoea, the breathing becomes very rapid and exaggerated, air enters freely into the lungs, the range of movement becomes very much increased, the normal ratio of inspiration to expiration is maintained, and the muscles of forced

respiration are called into action. On auscultation the respiratory murmur is harsh and puerile, death occurs in a few minutes.

(b) The grave, the second division of the pulmonary artery is plugged, the same series of symptoms occur but more gradually and recovery may take place.

(c) The benign, minute infarctions occur in the finer ramifications of the pulmonary artery, there may be few signs and very little dyspnoea.

The dyspnoea in these cases is due to the circulation through the lungs being obstructed, a deficient supply of blood is returned to the left side of the heart, and so the respiratory centre does not get a sufficient supply.

With regard to fat emboli, a large number of cases have now been recorded. The origin of the fat is mostly from the medulla of bone in cases of fracture, sometimes in injury to subcutaneous adipose tissue in operations, and according to some authorities it occurs in diabetic coma, but I shall refer to this again.

Eberth considers that coagula in the heart some-

times undergo fatty degeneration and break down, giving rise to fat emboli.

When there is a large quantity of fat present in the blood, all the pulmonary capillaries get plugged and then severe dyspnoea and death occur. According to Professor Czerny there is first produced oedema of the lungs and then carbonic acid poisoning; but there is no doubt that the symptoms are due to a deficient supply of arterial blood to the brain and tissues generally, much more than to the accumulation of carbonic acid in the blood.

In air embolism the air enters the veins during some operation on the large veins. The pressure in the large veins is very low and during inspiration there is even a degree of suction, so that if a vein be opened during inspiration air enters.

A considerable amount of air is necessary to produce any result, Blundell injected five drachms of air into the jugular vein of a small dog without producing any effect. Hutchinson injected a large quantity of air into a horse's jugular vein and caused death, he found the right side of the heart

distended like a bladder; this, together with the fact that repeated injections of small quantities of air into the circulation does not produce any serious results, points to the conclusion that the dyspnoea and death are not due to embolism, but probably to the right side of the heart becoming distended with air. This prevents the admission of blood from the venae cavae, and the right ventricle in contracting merely squeezes together the elastic air which expands again during diastole and so the distribution of blood ceases and death occurs.

III. Dyspnoea due to an abnormal state of the blood.

(1) Anaemia of all varieties. Here the dyspnoea is due principally to a deficient supply of oxygen to the respiratory centre, owing to the diminution of the oxygen carrier, Haemoglobin, in the blood; I say principally, because another factor often in operation is the dilatation of the heart which so often accompanies anaemia and interferes with efficient circulation.

As the cause of the dyspnoea is of gradual

onset, the dyspnoea usually only occurs on exertion and its characters are similar to those of dyspnoea from exertion described below.

(2) Dyspnoea due to an impure altered state of the blood other than due to simple diminution of its oxygen carrying elements. The best and most easily studied variety in this class is the dyspnoea produced by violent exertion. Its chief characteristics are - a great desire for air ('besoin de respirer'), a sensation of a fulness in the head and throat and distension of the chest, there is a peculiar feeling under the sternum like the sense of heat. The rate of breathing is greatly increased, it may be up to 50. or 60. per minute, the range of movement is increased, the breathing is deep, and three or four times the usual quantity of air is changed at each respiration: The normal ratio of inspiration and expiration is altered, both are very much shortened, but expiration much more so than inspiration and both become about equal in length of time. The active moving powers are altered, the forced muscles of respiration being

called into action, so that we find all the conditions of normal respiration changed.

The cause of the dyspnoea of exertion is not a deficiency of oxygen, for although there is an increase in the amount of oxygen used, the rapid breathing makes up for this and the blood is kept well oxygenated and rapidly distributed over the body. The real cause is that the rapid muscular contractions lead to an accumulation of carbonic acid and other waste products, such as sarcolactic acid, in the blood, which stimulate the respiratory centre. An additional cause may be the increased temperature of the blood caused by the muscular contractions.

Bonnal of Nice made numerous observations on the effect of muscular exercise on the body temperature, he found that all muscular exercise, even if of short duration, caused invariably a rise of temperature, rarely however to a point exceeding 101°F. the greatest elevation he observed was to 103.2° F. in a celebrated runner who had just run eleven miles in an hour and a half.

Heat or Thermal Dyspnoea.

It has long been observed that in all conditions in which the temperature of the body is raised, the rate of breathing is increased. It is extremely difficult to determine the exact manner in which the elevation of temperature acts in producing dyspnoea, whether through the cerebral convolutions first and then through them on the respiratory centre, or on the skin and through the cutaneous nerves, or through the pulmonary branches of the vagi, or directly on the respiratory centre.

Goldstein made a number of experiments on animals and came to the conclusion that the increased temperature did not act through the skin because in heated animals cold effusion did not at once stop the dyspnoea, also that it did not act through the cerebrum or owing to any feeling of pain or uneasiness in the animal because it was not prevented by the injection of morphia, that it did not act through the pulmonary branches of the vagi as it occurred after they were divided.

He placed the exposed carotid of a dog on a

heated brass tube and as dyspnoea then occurred he came to the conclusion that it was produced by the action of the heated blood on the respiratory centre.

Sihler found that in Goldstein's experiment of laying the exposed carotid on a heated brass tube, the dyspnoea was produced just the same if the artery were clamped higher up, and so he maintained that the dyspnoea was due to pain, and that when the blood really is heated the dyspnoea is due principally to skin stimulation and also partly to increased venosity of the blood, the pyraexia increasing the chemical changes in the body. He succeeded in producing apnoea by artificial respiration in heated animals after cutting off the skin stimuli by dividing the spinal cord, He also found that in dogs exposed to a heated atmosphere dyspnoea occurred before their temperature was raised.

M. Mertchinsky made a series of experiments on rabbits, and he agrees with Goldstein in believing that heat dyspnoea is produced by the direct action of the heated blood on the respiratory centre, he

observed that the type of heat dyspnoea was quite different from that due to increased venosity of the blood. In heat dyspnoea the respirations are short and shallow, whilst in asphyctic dyspnoea they are deep and full.

These observations, although of extreme interest, are of little or no practical value, as, clinically, we never see a case of pure heat dyspnoea, that is dyspnoea produced by an increased temperature of the blood or body, without other factors, acting on the respiratory function. We know that in fever of every kind we have a greatly increased tissue metabolism and so a correspondingly increased venosity of the blood, and this is the main factor in causing dyspnoea, there are also various ptomaines &c. in the blood which probably directly stimulate the respiratory centre, or else diminish the available amount of oxygen in the blood by combining with it, or by reacting on the red blood corpuscles in such a manner as to prevent them taking up the usual amount of oxygen. This form of dyspnoea occurs in all diseases in which there is

pyraexia, I have already stated why I believe pneumonia to come under this class.

In Brights disease in any of its forms we frequently meet with dyspnoea, which may be due to many causes, such as anaemia, pleural effusion, oedema of lungs, weak heart &c. but apart from these, dyspnoea occurs which is dependant on the condition of the blood. It is of two kinds, continuous and paroxysmal, the latter has been termed renal asthma and is much the commonest variety, it usually occurs in the evening and may be quite absent during the day. The breathing is laboured, the range of movement very much increased, the rate of breathing is very much quickened, the ratio of inspiration to expiration is altered, both being shortened, but expiration much more than inspiration, the forced muscles of respiration are called into action, so it will be seen that it exactly resembles the dyspnoea of violent exertion; on auscultation the breathing is loud and puerile.

The cause of renal asthma is doubtful, Sir George Johnston believes it to be due to interrupted

circulation through the pulmonary capillaries, due to spasm of the pulmonary arterioles which are stimulated to this excessive contraction by the influence of impure blood on the vaso motor nerves and centre. In support of this he says that the panting laborious breathing is exactly like that in pulmonary embolism. Dr. Carter favours this view and points out the similarity between renal asthma and poisoning with muscarin. Lauder Brunton has shown that in poisoning with muscarin the dyspnoea which is produced is due to spasm of the pulmonary and other arterioles, Dr Carter suggests the possibility of neurine and choline being in the blood in uraemia and causing dyspnoea. These bodies have been separated from the human tissues by Brieger and they are structurally and morphologically allied to muscarin.

The other view is that the dyspnoea is due to the action of a certain substance or substances in the blood which stimulate the respiratory centre directly to increased action, or may possibly interfere with the due interchange of oxygen and carbonic

acid between the blood and the tissues.

In diabetic coma dyspnoea frequently occurs, it comes on suddenly and with violence, both expiration and inspiration are prolonged and deep, the air passes freely into the lungs but apparently does not oxygenate the blood.

There are two theories as to the cause of the dyspnoea, (1) Fat embolism (2) The presence of acetone in the blood. It has long been known that in diabetes there is an excess of fat in the blood.

Saunders and Hamilton in 1879. described a case in which there was intense dyspnoea'with inspiration and expiration of extraordinary fulness and depth like a man who had won a mile race', it presented a typical picture of air hunger, at the post mortem examination fat emboli were found in the lungs and other organs. They believed the symptoms were produced by the fat embolism causing insufficient oxygenation of the blood, carbonic acid poisoning and coma with dyspnoea.

Starr and Hertz have also recorded cases in

which fat emboli were found after death.

Professor Fraser recorded a case in 1882 in which there was marked dyspnoea and coma, on drawing a drop of blood from the finger it was of a creamy pink colour and full of oil globules, at post mortem examination numerous fat emboli were found. Saundby and Foster believe that dyspnoea and coma are due to acetonaemia. On mixing acetone with blood they produced a creamy liquid just like the blood in diabetic coma, the creamy colour was due to the presence of a large quantity of molecular matter which looked like fat globules but it was not dissolved out by ether, this however does not go any way towards disproving the fat embolism theory, as in diabetic coma it has undoubtedly been proved that the blood contains oil globules. At post mortem examinations there has been found fat embolism, but there have been many cases in which there was typical dyspnoea and coma and in which the most careful examination after death failed to find any fat embolism, Dr. Stephen Mackenzie has recorded eighteen such cases.

Mansell Moullin believes that in fractures as a rule there is fat embolism and yet as a rule there is no dyspnoea or coma.

No doubt fat embolism when it occurs increases the dyspnoea and coma, but probably the essential cause is the presence in the blood of acetone or an acetone producing substance such as aceto-acetic acid which by its toxic effects on the nerve centres produces dyspnoea and coma.

Dyspnoea due to poisoning with certain gases. (1) Those which displace the oxygen and form a permanent stable compound with the Haemoglobin. Carbon monoxide when respired has been proved by Hoppe-Seyler, Lather Mayer and Claude Bernard to displace the oxygen and form an intimate combination with the haemoglobin in the blood and prevent it from taking up oxygen, hence the dyspnoea and death from asphyxia.

Pettenkofer has shown that in poisoning by coal gas the toxic effects are due to the carbon monoxide which it contains.

In Hydrocyanic acid poisoning death is extremely

rapid, preceeded by convulsions which were believed to be due to suffocation, but Lauder Brunton has observed that the convulsions come on before the mucous membranes become at all cyanosed and therefore are not due to a venous state of the blood. He believes the dyspnoea and convulsions caused by hydrocyanic acid to be due to its action on the cells of the medulla itself by which it prevents ordinary internal respiration taking place in them, it does not displace the oxygen in the blood like carbon monoxide or lock it up in the form of methaemoglobin like the nitrites.

(2) Narcotic gases, Carbonic acid when inhaled in considerable quantity, (about 10 per cent in the air even though oxygen be present in sufficient quantity) produces dyspnoea. Respiration becomes rapid and deep, the inspiratory and expiratory movements being increased, the ordinary interchange between carbonic acid in the blood and oxygen in the air is prevented, carbonic acid accumulates in the blood, the process of oxidation is interfered with and dyspnoea occurs.

Nitrous oxide when inhaled pure, among its other effects produces dyspnoea by depriving the respiratory centre of oxygen.

(3) Reducing gases. Sulphuretted hydrogen produces dyspnoea and death from asphyxia by reducing and then decomposing the haemaglobin in the blood, it robs the red cells of oxygen, forming sulphur and water (Hoppe-Seyler). Accidents from this are met with among workmen cleaning out cesspools where sulphuretted hydrogen accumulates. Phospuretted hydrogen acts in the same way, forming phosphoric acid and water. Arsenuretted hydrogen and Antimoniuretted hydrogen also act in the same way, but in addition haemoglobin passes out of the stroma and appears in the urine. Cyanogen absorbs oxygen and decomposes the blood.

Certain drugs stimulate the respiratory centre directly and increase its activity, the respirations become quicker and deeper so that more respiratory work is done. The following drugs act in this way:- Strychnine, Ammonia, Atropine, Duboisine, Brucine, Thebaine, Apomorphine, Emetine, Members of the Digi-

Digitalis group, Salts of Zinc and copper. Many other drugs produce dyspnoea indirectly, such as pilocarpine, which probably acts by weakening the heart's action.

IV. Dyspnoea of Central nerve origin.

In cerebral disease of almost any kind, such as tumour, haemorrhage, meningitis, there may occur dyspnoea of an irregular type, as previously mentioned it may assume the Cheyne-Stokes type, it is frequently sighing and irregular, it depends upon irritation of the respiratory centre or of fibres closely connected with it.

In connection with hysteria there is sometimes the most marked dyspnoea, Mackey relates a case in which the respirations went up to 128 per minute.

A distinguishing point is that the pulse is always slow no matter how great the dyspnoea when it is of hysterical origin.

It is sufficient to mention that dyspnoea occurs from emotion and also in connection with chorea.

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